Addiction

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DIAGNOSIS

Substance *Abuse*

DSM-IV-TR Criteria for Abuse

a maladaptive pattern of substance use leading to clinically significant impairment or distress as manifested by 1 (or more) of the following occurring within a 12 month period

- recurrent substance use resulting in a failure to fulfill major role obligations at work, school or home
- recurrent substance use in situations where it is physically hazardous
- recurrent substance-related legal problems
- continued use despite persistent social or interpersonal problems caused or exacerbated by the substance

Symptoms have never met the criteria for substance dependence

Substance *Dependence*

DSM-IV-TR Criteria for Dependence

a maladaptive pattern of substance use leading to clinically significant impairment or distress as manifested by 3 (or more) of the following occurring within a 12 month period

- tolerance
- withdrawal
- larger amounts or longer periods than intended
- persistent desire or unsuccessful efforts to cut down or control
- great deal of time spent in activities to obtain substance
- social, occupational or recreational activities given up
- continued use despite knowledge that physical / psychological problems will continue or be made worse

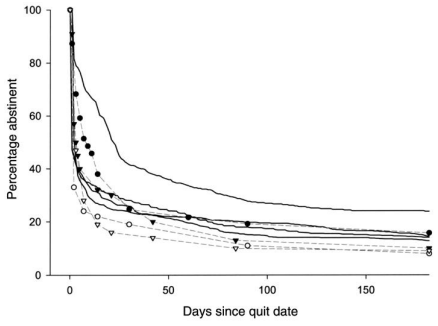
On the horizon? Likely changes for DSM-V (O'Brien, 2011)

Use term 'addiction' rather than dependence
- Psychological vs. physiological
- Non-substance addictions

Add 'craving'

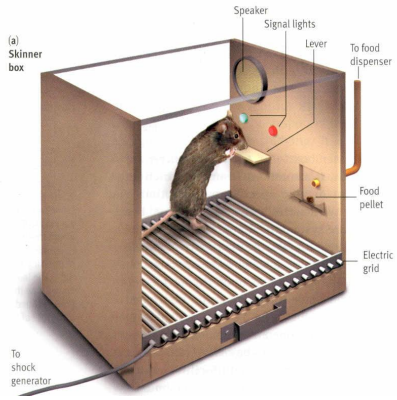
Drop distinction between 'abuse' and 'dependence'
- Disorders on a continuum

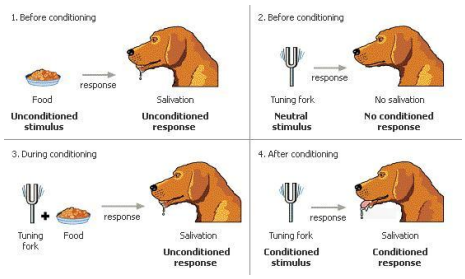
The problem of relapse (e.g. smokers; Hughes, 2004)



Theories

Conditioning
Choice
'Disease' model
Opponent processes / dysregulation
Incentive learning
Habit
Cognitive processes
Dual-process cognitive models
Impulsivity / executive dysfunction





Operant and classical conditioning

Operant conditioning: R-O (Response Outcome) learning
- E.g. drinking alcohol results in pleasant feelings
- Outcome of a response influences likelihood of repeating that response in future
- Positive and negative reinforcement; punishment

Classical conditioning: S-O (Stimulus Outcome) learning
- E.g. sight and smell of beer is paired with effects of alcohol
- After many pairings, the sight of beer(S) leads to a conditioned response (e.g. craving) (CR) because it is paired with alcohol(O)

CHOICE (VERSUS COMPULSION)

Heavy drug use (addiction?) as choice

Drug use as cost-benefit analysis (E.g. Becker & Murphy, 1988; Bigelow, 1998).

Benefits (e.g. high (cocaine, heroin), increased alertness (nicotine), social aspects (alcohol, nicotine) outweigh costs (losing your job, illness, an early death)

Any consequences of drug use are perceived as improbable or likely to occur far in the future

If adverse consequences do occur (e.g. emphysema, incarceration), then it is too late to stop?

Evidence in support

Drugs (particularly opiates / psychostimulants) produce positive effects, therefore they function as positive reinforcers and are 'attractive'.

Reinforcement of drug abstinence reduces drug-taking (e.g, voucher therapy, behaviourally-contingent pharmacotherapy).

The motivation to abstain is associated with abstinence.

Problems with straightforward choice accounts

As frequency of drug use increases:

- Negative consequences (including withdrawal; missed work etc.) increase
- Positive consequences (e.g. euphoria) often decrease due to tolerance.

So drug use should naturally 'extinguish' once addiction develops?

Why not?

Heyman's (1996) view

Each time we consume a commodity (e.g. a Chinese meal) the subjective utility of that commodity decreases.

- Addictive drugs are no different: so each time a drug is used, its utility is reduced

But each time a drug is used, the utility of competing activities (e.g. doing some exercise, doing some work) also decreases, *and to a greater extent than the utility of drug use*.

- Supported by demonstrations of 'anhedonia' in addiction, e.g. Powell et al (2004)

So drug use is always an attractive option – a way of maximising utility. In addiction, even though its absolute utility has increased, its relative utility (to other activities) has increased.

- Becker & Murphy made similar predictions: i.e. once the drug user is in a state of drug-induced distress, the utility of competing activities drops: drug use is the only activity left with any remaining utility.

Different perspectives on 'choice'

The *observer* sees the life that the addict leads (e.g. homelessness, poverty, ill health) and can easily imagine a 'better', drug-free life.

But the *drug user* perceives a drug-free life as lacking in any pleasure or escape that they get from drugs

- E.g. a home, a good job, or improved health may not automatically occur if drug use ceases
- Delay to receipt of these benefits: temporal discounting (more on this later).

The drug user's view of a drug-free life....

Choose life. Choose a job. Choose a career. Choose a family. Choose a fucking big television, Choose washing machines, cars, compact disc players, and electrical tin openers. Choose good health, low cholesterol and dental insurance. Choose fixed- interest mortgage repayments. Choose a starter home. Choose your friends. Choose leisure wear and matching luggage. Choose a three piece suite on hire purchase in a range of fucking fabrics. Choose DIY and wondering who you are on a Sunday morning. Choose sitting on that couch watching mind-numbing sprit- crushing game shows, stuffing fucking junk food into your mouth. Choose rotting away at the end of it all, pishing you last in a miserable home, nothing more than an embarrassment to the selfish, fucked-up brats you have spawned to replace yourself. Choose your future. Choose life...

Benefits of using drugs

I chose not to choose life: I chose something else. And the reasons? There are no reasons. Who need reasons when you've got heroin?

People think it's all about misery and desperation and death and all that shite, which is not to be ignored, but what they forget is the pleasure of it. Otherwise we wouldn't do it. After all, we're not fucking stupid. At least, we're not that fucking stupid. Take the best orgasm you ever had, multiply it by a thousand and you're still nowhere near it. When you're on junk you have only one worry: scoring. When you're off it you are suddenly obliged to worry about all sorts of other shite. Got no money: can't get pished. Got money: drinking too much. Can't get a bird: no chance of a ride. Got a bird: too much hassle. You have to worry about bills, about food, about some football team that never fucking wins, about human relationships and all the things that really don't matter when you've got a sincere and truthful junk habit.

Why the dissociation between behaviour (drug use) and intentions (to stop)?

Different ways of asking about future drug use intentions:

'Do you want to stop using heroin' (answer YES)

'Does the prospect of a becoming an ex-heroin user, with all that that entails, appeal to you more than the prospect of continuing as a heroin user' (answer ERRRMMMMM……)

Ties in to John Davies' "myth of addiction" concept

The myth of addiction (Davies, 1992)

Drug users put their drug use down to **choice** when they are with their peers, but they put it down to '**addiction**' when they are with the police, doctors etc.

Why? Minimise blame and personal responsibility, increase chances of receiving help and medication.

Also serves the needs of:

- The medical profession
- Researchers (like me!)
- Families of addicts (no need to take responsibility or 'blame' the addict)
- Politicians (if addiction is not a medical problem, it must be a kind of 'moral' one)

BUT: doesn't this overlook the role of compulsion / craving?

What is choice?

Choices only occur when people consciously consider alternatives.

- Automaticity and behaviour in general and drug-taking in particular (e.g. Tiffany, 1990)

The 'choice' must be made in the absence of competing drives

- E.g. if someone points a gun to my head and instructs me to drink whisky, do I really have a choice?
  - Is the experience of a powerful craving the same as this? COMPULSION

If drug users want to exercise restraint, yet they continue to use drugs, how was their behaviour a consequence of their 'choice'?

- …back to the issue of whether drug users really want to stop drug-taking (or not)

Irrational, ill-informed choices with unstable preferences

Unstable preferences: e.g. **choose** to abstain when in treatment, but change your mind and **choose** to use drugs again when back in the home environment

- Difficult to test?
- Relies on post-hoc explanation of the causes of behaviour?
- If choices are 'governed by strong appetites' (Skog, 2000), in what sense are they choices?

Ill-informed choices: e.g. addicts tend to underestimate likelihood and severity of drug-related diseases (e.g. emphysema), while overestimating their own ability to quit.

- But some evidence that smokers are MORE aware of negative consequences of smoking than non-smokers!

Skog's (2000) view of unstable preferences

Drug users *can* abstain (cf. Bigelow)– it's just that their preferences change, and they choose not to.

- Motivational ambivalence
- Unstable preferences give only the *appearance* of loss of control.

To treat addiction, we just need to increase costs of using, and benefits of not using (e.g. token economies)

Again: choice must be involved to a degree, even if that choice is highly constrained, and influenced by many different factors.

THE 'DISEASE' MODEL

'Alcoholism' as a disease (Jellinek, 1960)

IRREVERSIBLE ('you can go from a cucumber to a gherkin but you can't go from a gherkin back to a cucumber')
PROGRESSIVE
INCURABLE (always 'recovering' never 'recovered')

Characteristics of the model: Inability to control drinking
Goals of treatment: Long term abstinence

Disease model

Biological predisposition

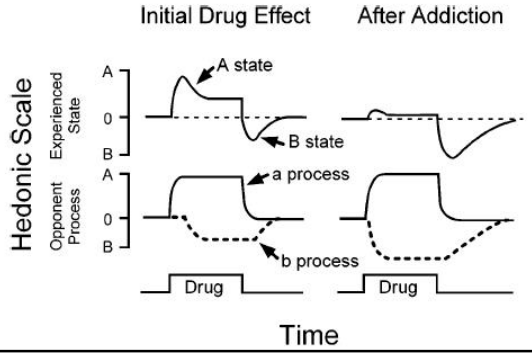
Withdrawal relief and drug 'priming' effects are crucial

Underlying principles in AA & NA and the Minnesota Model

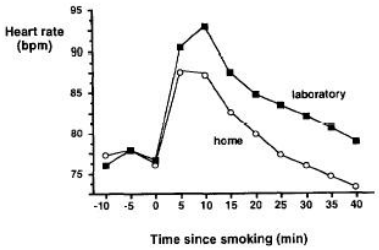
12 step programme

OPPONENT PROCESSES / HEDONIC HOMEOSTATIC DYSREGULATION

Solomon & Corbit (1973)



Conditioned tolerance (Mucha et al. 1996)



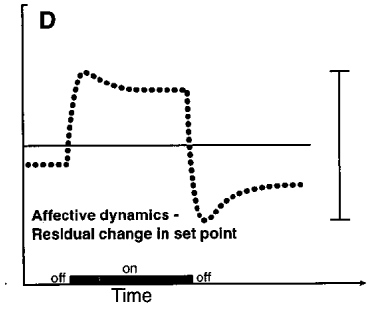
Hedonic homeostatic dysregulation (Koob& Le Moal, 1997)

> Allostasis > individual maintained at a new ('low') set-point.

Sensitization and counteradaptation processes cause further attempts to maintain mood at this new set point > but with increasing drug use, it becomes increasingly difficult to maintain this set point.

At some point, allostasis breaks down and the individual cannot maintain a set point > 'spiralling distress', or mood disturbance.

Hedonic homeostatic dysregulation (e.g. Koob & Le Moal, 1997)



REWARD AND INCENTIVE LEARNING

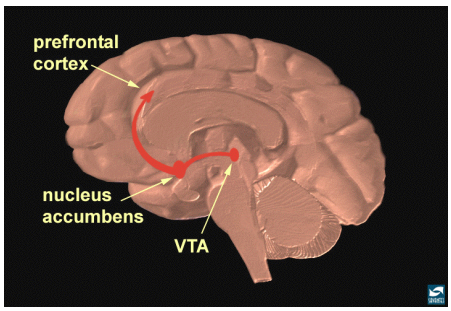
"Reward" systems in the brain

Mesolimbic dopamine system: the *nucleus accumbens* and areas that project to and from it.

All drugs of abuse stimulate dopamine release in this area (directly or indirectly).

Also stimulated by food, sex, warmth, and other "natural" rewards.

Is this the 'pleasure centre'?



Nucleus accumbens dopamine and conditioning (White, 1996)

CS that predicts a rewarding US activates the nucleus accumbens in both animals and humans (Schultz, 1998).

Activity often greater to the CS than to the US (Schultz, 1998)

Dopamine antagonists in nucleus accumbens can block conditioning.

Implication: involved in signalling of reward?

If drugs release dopamine in this area > signal 'salience', promote conditioning?

- > Very fast / effective learning about drug cues, could explain cue reactivity?

The nucleus accumbens is not just the 'pleasure centre' (Spanagel & Weiss, 1999)

The dopamine response…

- Habituates quickly (**for natural rewards**)
- Is strongest for 'novel' stimulation (e.g. novel foods, drugs).
- …so response diminishes for 'predictable' food.
- Is often stronger in response to a CS that signals a US, than to the US itself
- Is seen for aversive stimuli (e.g. shock)

Not really compatible with the notion that nucleus accumbens dopamine = pleasure

Nucleus accumbens dopamine codes for 'salience'

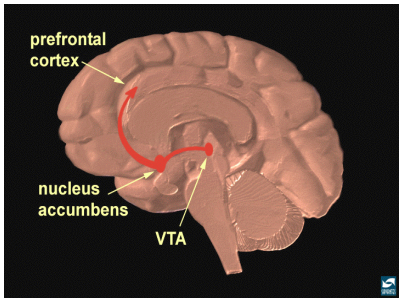
Drugs which increase dopamine activity increase the 'salience' of environmental cues

- E.g. Volkow et al. (2002) – methylphenidate increases interest in and desire for food in response to food cues.
- Effect correlated with extent of dopamine release in nucleus accumbens

Implication – the nucleus accumbens is involved in **incentive learning?**

- Perhaps the role of this system is to learn about what environmental stimuli signal 'rewards'?

So this system signals the *significance* or *salience* of rewarding events, but is not just involved in 'pleasure' (?)



Interim summary

Dopamine in the nucleus accumbens is involved in….

The 'rewarding' and 'reinforcing' effects of drugs of abuse and 'natural' rewards.

- But responses to CS are stronger than to US
- Also responds to aversive stimuli

Learning (conditioning) about which cues predict the occurrence of rewards / reinforcers (incentive learning).

The recognition of significant (**salient**) cues in the environment (i.e. **conditioned stimuli** paired with natural and drug rewards)

If drugs of abuse and 'natural' rewards all stimulate the same circuitry, why are we more likely to get addicted to drugs than to water?

Larger dopaminergic response to drugs?

- No

Differences in habituation / sensitization?

- Dopaminergic response to 'natural' rewards habituates over time
- But the same is not seen with drug rewards
- In fact, the dopaminergic response to drug rewards may **sensitize** over time.

Differences between drug and non-drug reinforcement – sensitization (Robinson & Berridge, 1993, 2000)

Behavioural activation response to drugs of abuse **sensitizes** with repeated administration – **assumed** to be mediated by sensitization of dopamine in nucleus accumbens (Wise & Bozarth, 1987).

Only with spaced (not continuous) presentation of the drug

- Human drug-taking?

Contextually mediated – effects strongest in drug-paired context.

- Explains power of drug-related cues?

Why does the brain respond differently to drug and 'natural' rewards?

Brain likely evolved to respond to 'natural' rewards – nucleus accumbens and related dopaminergic circuitry exist to register reward and to learn about environmental cues that predict 'good' events (e.g. food).

But drugs of abuse share the ability to activate this region and their long-term effect is different from natural rewards

- Why? Probably just a fluke? Drugs of abuse represent tiny proportion of substances that we ingest.

Drugs of abuse 'hijack' this area of the brain / dopaminergic system > they seem excessively 'rewarding' and make reward cues seem excessively 'salient'.

Evidence for dopaminergic sensitization?

Direct evidence: Nestby et al. (1997) – dopaminergic response to alcohol, cocaine, and morphine in the nucleus accumbens sensitizes after repeated administrations.

But controversial – evidence for dopaminergic sensitization is not that compelling!

Sensitization in humans

Behavioural sensitization

- Laboratory studies: mood / activity and cocaine (Strakowski et al., 1996)
- But not shown with other drugs

Dopaminergic sensitization:

Little evidence

- Difficult to study; need to compare **response to drug** in addicts and drug-naïve volunteers > ethics?
- But in fact, cocaine addicts show dampened dopamine response to methylphenidate compared to controls (Volkow et al., 2004) (tolerance?)

Summary – incentive-sensitization model

The notion that craving becomes excessive, and drug-related cues are able to control behaviour, is consistent with evidence and observations of dependent individuals.

This model can also explain the dissociation between 'wanting' drugs and 'liking' their effects

But the role of dopamine sensitization in these effects is still a matter of controversy.

HABIT

Tiffany's (1990; 1998; 2000) model

Drug use is automatic

Craving is non-automatic: it occurs when drug use is impeded in some way.

- Abstinence-promotion
- Abstinence-avoidance

Is drug use 'automatic'?

Compulsive drug use

1. Loss of control
2. Stimulus bound
3. Difficult to regulate
4. Stereotyped patterns of use

Automatised performance

1. Lack of control
2. Stimulus bound
3. Effortless
4. Rapid, stereotyped performance
5. Lack of awareness

Think of riding a bike, seatbelt use

Evaluation of Tiffany's model

Theory can account for 'absentminded' relapse, and it has intuitive appeal for certain addictions (e.g. smoking).

- E.g. Catley et al. (2000) – 6% of smoking lapses were 'absentminded', although background craving levels were not minimal.
- These were smokers attempting to quit – perhaps higher % in smokers not attempting to quit?

But is compulsive drug use really the same as riding a bike?

Habitual responding

S > O > R

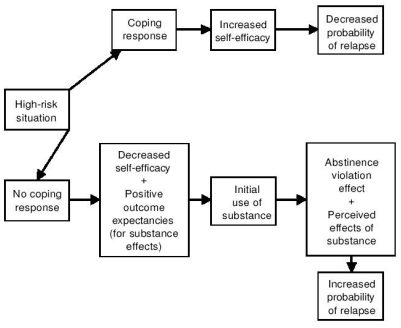
Glass of beer > Feel good > Consume

After many drinks S > R

Drink > Consume (anticipated outcome no longer important)

COGNITIVE PROCESSES

Marlatt (1996)



Outcome expectancies (Jones et al., 2001)

Long-term memory structures about R-O relationships.

AEQ item:

'When I'm antisocial, drinking makes me more gregarious' AGREE / DISAGREE

Also for smoking, cannabis….

Social learning theory: R-O beliefs mediate behaviour (> drug use)?

Types of expectancies

Positive: Social facilitation vs. tension reduction
- Maps to positive and negative reinforcement?

Negative: e.g. alcohol makes people violent

Greater endorsement of positive **and** negative expectancies in smokers compared to non-smokers (Litz et al., 1999)
- Why problematic?

Outcome expectancies and alcohol use

Alcohol consumption positively correlated with positive expectancies, negatively correlated with negative expectancies (e.g. Brown et al., 1987).
- But % variance explained is modest when age, gender, past drinking are controlled.

Positive expectancies control initiation, negative expectancies control cessation / control over excessive drinking (McMahon et al., 1994).

Cause or consequence?

AOEs precede and predict alcohol consumption in children (Christiansen et al. 1989).

AOEs predict prospective alcohol dependence symptoms (Kilbey et al., 1998), and relapse risk (Brown, 1985).

Priming positive expectancies increases alcohol consumption (Carter et al., 1998)

Expectancy challenge as prevention? Inconsistent and unimpressive results.

Expectancy challenge and alcohol consumption (Darkes & Goldman 1993).
- But failures to replicate
- Effects maintained long-term?

Self-efficacy

Confidence in ability to abstain

Popular belief that high self-efficacy is predictive of treatment success (and some evidence)

But even this is not as robust as is generally assumed (abstainers vs. lapsers) (Gwaltney et al., 2009)

DUAL-PROCESS MODELS: IMPLICIT / AUTOMATIC COGNITIVE PROCESSES

Wiers et al. (2007)

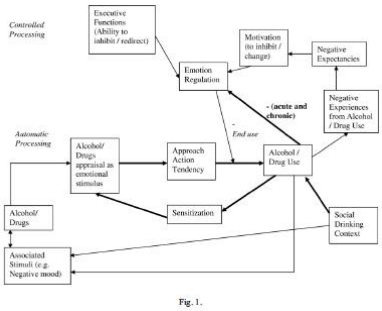


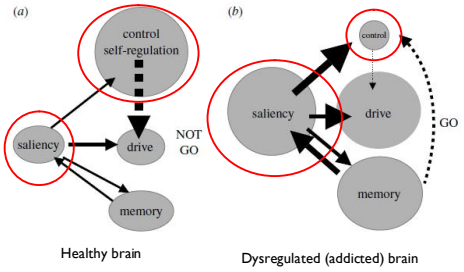
Fig. 1.

Examples of implicit / automatic processes

- Attentional bias
  - Predicts relapse / prospective drug use (Cox et al., 2002; Fadardi& Cox, 2009)
- Automatic associations
- Approach tendencies
  - Predicts drug use (Wiers et al., 2009)

IMPULSIVITY / EXECUTIVE DYSFUNCTION

Volkow et al. (2008)



Evidence

- Substance users more 'impulsive' than controls on self-report and behavioural measures, e.g. response inhibition, delay discounting (Verdejo-Garcia et al., 2008)
- Risk-factor: pre-morbid? (Giancola & Tarter, 1999)
- Worsen with prolonged heavy drug use? (Jentsch& Taylor, 1999).

Part 2

KEY CONCEPTS AND PROCESSES IN ADDICTION AND RELAPSE

Reinforcement and choice

- Drug use is positively (e.g. pleasure) and negatively (e.g. withdrawal relief, tension reduction) reinforced.
- Closely related to the notion of drug use as 'choice'

Cues, automaticity, and implicit processes

- Drug-related cues are paired with drug effects
- Those cues elicit physiological changes and subjective craving
- Over time, those cues might elicit drug-seeking behaviour automatically ('habit')

Tolerance / physiological adaptations

- With repeated drug use, tolerance develops; context-dependent
- May be associated with an adaptive, 'drug-opposite', negative emotional state; a change in set-point

Motivation and salience

- Drugs 'hijack' brain areas involved in attribution of salience
- Results in excessive craving for the drug and increased 'importance' attached to drugs and drug cues

Impulsivity and executive control

- The previous processes all increase the drive to use drugs
- Exacerbated by impaired control over drug use, which may be pre-morbid, but may get worse with chronic drug use